pesticide sprayers. So administration of supplementary nutrients or improving defense system in these subjects is advised.

Keywords: Hypothyroidism, Pesticide, TSH, Immunoassay

doi:10.1016/j.clinbiochem.2011.08.1063

Oral – [A-10-831-1]
Protective role of N-acetyl-cysteine on diazinon induced oxidative stress in rat kidney

Farideh Izadi[a], Mahvash Jafari[b], Alireza Asgari[b], Maryam Salehi[b]
[a]*Department of Biology, Islamic Azad University, Tehran, Iran*
[b]*Baqiatall University, Iran*
E-mail addresses: Izadi_FArideh@yahoo.com (F. Izadi), jafari@bmsu.ac.ir (M. Jafari)

Introduction: Organophosphate pesticides (OP) may cause generation of reactive oxygen species (ROS), which may lead to oxidative stress. Diazinon (DZN) is an organophosphorus insecticide that has been widely used throughout the world with applications in agriculture and horticulture for controlling insects. N-acetyl-l-cysteine (NAC), precursor of reduced glutathione (GSH), is an antioxidant and free-radical scavenger. In this study, the possible protective role of NAC in DZN-induced toxicity was evaluated in male Wistar rats.

Materials and methods: In vivo experimental groups were: control group (corn oil as diazinon solvent), DZN group (100 mg/kg, i.p.), NAC (160 mg/kg, i.p.) group and NAC–DZN group. 24 hours after injection, animal to ether anesthesia and kidney removed and superoxide dismutase (SOD) and catalase (CAT) activities, as well as GSH and malondialdehyde (MDA) levels were determined by biochemical methods.

Results: The result showed that SOD and CAT activities and MDA level were increased, while GSH level was decreased in DZN treated rats as compared to control. NAC–DZN groups were found to improve these disorders.

Conclusion: The results suggest that NAC provides protection against DZN-induced oxidative stress and prevents lipid peroxidation in kidney.

Keywords: Diazinon, N-acetyl-cysteine, Antioxidant system, Rat, Kidney

doi:10.1016/j.clinbiochem.2011.08.1064

Oral – [A-10-1001-1]
Activation of Nuclear Factor-kappaB by doxorubicin leads to inhibition of extrinsic pathway of apoptosis in H9c2 cells

Leila Hosseinzadeh, Gholamreza Karimi,
Fatemeh Mosaffa, Mitra Rezaee Asgharian
Faculty of Pharmacy, Kermanshah University of Medical Sciences, Kermanshah, Iran
E-mail addresses: L_hoseinzadeh@yahoo.com (L. Hosseinzadeh), gkarimi@mums.ac.ir (G. Karimi), ft_mosaffa@yahoo.com (F. Mosaffa)

Introduction: Doxorubicin (DOX), a chemotherapeutic agent, can give rise to serve cardiotoxicity by inducing apoptosis. The exact mechanism of DOX-induced apoptosis has not been fully understood yet. Here, we evaluated the effect of DOX on some important genes involved in apoptosis and also Nuclear Factor-kappaB (NF-κB) activity in H9c2 cardiomyoblast cells.

Materials and methods: Cell viability was determined by MTT. Apoptosis was assessed by annexine V/PI double staining. Real time RT-PCR was used to evaluate the expression of genes. The DNA-binding capacity of NF-κB was examined by ELISA.

Results: DOX-mediated cytotoxiocity is executed by inducing apoptotic cell death. DOX induced apoptosis by down regulating anti-apoptotic Bcl-2 and up-regulating pro-apoptotic Bax. Consequently, the ratio of Bax to Bcl-2 is significantly increased upon treatment with DOX leading to increase caspse-9, the initiator caspase of mitochondrial pathway for apoptosis, expression. However, it had no effect on the expression of caspase-8 the mediator of extrinsic pathway. DOX wasn't able to inhibit expression of anti-apoptotic gen cIAP1. Also, we observed that NF-κB activity was sharply increased by incubation in the presence of DOX. Previous studies have shown that depending on the cell models, apoptosis from DOX can follow different pathways. Our findings demonstrated that apoptosis induced by doxorubicin occurred through intrinsic pathway rather than by extrinsic pathway. It was indicated that NF-κB induces multiple factors to regulate apoptosis including cIAP1.

Conclusion: Based on these results, it is possible that NF-κB activation suppresses extrinsic pathway of apoptosis through blocking the cIAP1 down-regulation and consequently caspase-8 activation by DOX in H9c2 cells.

Keywords: Doxorubicin, NF-kappaB, Apoptosis, H9c2 cells

doi:10.1016/j.clinbiochem.2011.08.1065

Oral – [A-10-1026-2]
Apoptosis induction of methysulfonylmethane in human gastrointestinal cancer cell lines

Naser Jafari, Shahab Bohlooli
Ardabil University of Medical Sciences, Ardabil, Iran
E-mail address: n.jafari@arums.ac.ir (N. Jafari)

Introduction: Methylsulfonylmethane (MSM) is an organic sulfur containing compound that occurs naturally in a variety of fruits, vegetables and unpasteurized milk. It has been used as a supplement in the last two decades. Recently it has been suggested that MSM has clinical potential as a non-toxic agent effective against metastatic melanoma. Considering the preventive effects of this substance on tumor onset and nontoxic to healthy body we have investigated in vitro effects of methylsulfonylmethane on apoptosis induction in human gastrointestinal cancer cell lines.

Methods: The human cancer cell lines including, AGS, HepG2 and KYSE30 were cultured and incubated until confluence. The cells were removed and seeded in 96-well plates at a density of 1×103 cells/well and incubated overnight, and then treated with (21–30) mg/ml MSM. To stain apoptotic cells, the plates were centrifuged. The EB/AO dye mix was prepared in PBS and 10 μl was added to each well and cells were viewed and counted under an inverted fluorescence microscope.

Results: The results showed that after incubation at (21–29) mg/ml of MSM for 24 h the cells displayed a series of morphological changes including condensation and fragmentation of chromatin and nucleus, and formation of apoptotic bodies which were designated as typical evidence of apoptotic bodies. In contrast control cells exhibited a normal appearance. There was a significant increase of apoptotic cells in cancer cells after treating with MSM.

Conclusion: The results demonstrated that MSM exerts its anti-cancer and cytotoxic effect by inducing apoptotic cell death.

Keywords: Apoptosis, Methylsulfonylmethane, Cancer

doi:10.1016/j.clinbiochem.2011.08.1066